

March 18, 1875.

JOSEPH DALTON HOOKER, C.B., President, in the Chair.

The Presents received were laid on the table, and thanks ordered for them.

The following Papers were read :—

- I. "On the Behaviour of the Hearts of Mollusks under the influence of Electric Currents." By Dr. M. FOSTER, F.R.S., and A. G. DEW-SMITH, B.A., of Trinity College, Cambridge. Received February 1, 1875.

It has already been shown by Dr. Foster (Pflüger's 'Archiv,' vol. v. p. 191) that an interrupted current applied directly to the heart of the common snail (removed from the body) will cause an arrest of the rhythmic beat, the heart remaining *in diastole*.

The phenomena thus produced are altogether similar to those seen when the vertebrate heart is inhibited by stimulation of the pneumo-gastric nerve.

This inhibition by direct stimulation requires a current weaker than is sufficient to cause tetanus of the heart. It may be witnessed on either auricle or ventricle, and is the more easily obtained the more quickly the heart is beating. It takes place whatever be the position of the electrodes; and we find that it is possible thus to inhibit not only the whole ventricle but separate parts of a divided ventricle, provided these can be obtained, as they sometimes can, beating with a sufficiently rapid rhythm.

We have found no reason to think that there exists any localized inhibitory mechanism (corresponding to the sinus venosus ganglia of the frog's heart, for instance) any more than a localized automatic mechanism.

This inhibition by direct stimulation is, to say the least, an unusual fact; and the following inquiry was begun in the hope that further study of the heart of the same animal, and those of other mollusks, might bring to light some explanation of the matter.

We will relate first the results of our experiments with single induction-shocks and with the constant current on the snail's heart, and then pass on to the facts observed in other mollusks.

We have in all cases removed the heart from the body, and placed it in a watch-glass, in a little snail's blood. It will in this condition continue to beat with great regularity for hours.

Our reasons for always removing the heart from the body are as follows:—

The movements of the heart cannot be accurately observed *in situ* unless the overlying thin integument is divided and the pericardial chamber laid open. As soon as this is done the beats are seen to become very irregular. Every movement of the body influences the flow of blood to and from the heart, and every marked change in the flow affects the force and rhythm of the beat; hence observations like those we are about to record would be impossible with the heart *in situ*.

We have been unable to find any nerve in the body stimulation of which would inhibit the heart in the same way as stimulation of the pneumogastric does the vertebrate heart.

By removal of the heart from the body, therefore, we lose nothing essential.

A graphic record of each heart-beat would of course be impossible. In order to obtain a satisfactory register of our observations, we adopted the following plan. One of us acting as observer, and watching the heart through a microscope (50 to 100 diameters), signalled, by the sudden make and break of a constant current, each beat of ventricle or auricle, or of both. The make and break were recorded by a magnetic marker, as was also the application of the stimulus, on a travelling-paper, on which seconds were at the same time being marked. In this way a little practice enabled us to register with tolerable accuracy the rhythm of the beat, though we could not, of course, record the force or character of each pulsation.

Most of our observations were directed to the ventricle, and in our remarks we have only incidentally referred to the auricle.

We at first registered the auricular beats as well as the ventricular; but an increasing conviction of the independence (in an empty heart) of the movements of the two chambers, led us after a while to confine our attention to the ventricle.

The application of the current was made by one of us while the other, watching and recording the beats of the heart, was unaware of what was being done. The faithfulness of the observations was thus materially assisted.

Finding the heart of the common snail more vigorous, though smaller, than that of the edible snail, we confined our attention to the former.

The snail's heart consists, as is well known, of a thin-walled globular auricle, separated by a ring of non-contractile tissue from an oval, almost conical, fleshy ventricle. Into the former, opposite the auriculo-ventricular orifice, opens the large pulmonary vein. The latter at its apex, opposite the auricle, narrows to the large artery, which we have spoken of as the aorta. In speaking of the ventricle we shall frequently have occasion to distinguish the broader auricular end next to the auricle from the narrow aortic end, which is continuous with the aorta. In

removing the heart from the body, it is of importance, in order to secure a good rhythmic beat, that the incision should be carried through the aorta, and not invade the contractile tissue of the ventricle itself.

While the auricle is a sac, with quite thin and smooth walls, the bundles of fibres in the ventricular walls bulge out largely into the cavity, and are so arranged that the ventricle has the same spongy structure as that of the frog and many other animals.

Neither in the auricular nor ventricular wall can the presence of any nerve-cells, or collection of nerve-cells in the form of ganglia, be detected, whether in fresh specimens or in those treated with various reagents. The interlacing intricately arranged bundles of fibres are composed of a granular protoplasmic-looking tissue, quite unlike the ordinary muscular tissue of the body, and in many ways resembling the cardiac tissue of vertebrates.

The walls are thickly studded with nuclei, some of which possibly belong to an external tessellated epithelium. Other nuclei are undoubtedly the proper nuclei of the contractile elements, and the remainder seem to be of the nature of connective tissue.

Of none of them can it safely be said that they are the nuclei of nerve-cells. Molluscan nerve-fibres might undoubtedly, unlike vertebrate medullated nerve-fibres, easily escape detection; but Mr. A. S. Lea, of Trinity College, carefully examined for us the whole of both the auricle and ventricle without discovering any distinct nervous structures. He also went systematically over the margins of both the aortic and pulmonary orifices, but could find no nerves running into or out of the heart. In no other way could nerves become connected with the heart. And, opposed as it may seem to general experience, and still more to recognized opinions, we are led to the conclusion that the heart of the snail has no nervous connexion with the rest of the body; nay, more, that it has within itself no distinctly specialized nervous mechanism, but that its contractile elements are composed of protoplasm, arranged, it is true, more or less in fibres, yet otherwise but slightly advanced in differentiation.

The Effects of single Induction-shocks.

A single induction-shock sent through the ventricle of the snail's heart produces different results, according to the strength of the current employed. We have generally used the closing shock alone, or the opening shock alone, stopping the other shock in the usual way. Except that the opening shock acts, of course, as a stronger stimulus than the closing shock, we have not observed any marked difference between their effects.

When the shock is of a certain intensity (as, for instance, when a single Daniell's cell is used with the secondary coil of a Du Bois Reymond's induction-machine drawn out to 5 or 10), its application is immediately followed by a contraction. This contraction has all the characters of a normal beat, except perhaps that it is rather more sudden and of shorter

duration. Since the eye has been our only means of judging of the characters of the contraction, it being impossible to register the movements by the graphic method, we speak with great uncertainty on this point.

The stronger the shock the more marked, within certain limits, is the contraction. The effect, of course, in any particular case will depend not only upon the absolute strength of the current, but on its strength in relation to the irritability of the heart. Thus a heart with frequent and strong beats is affected by a weak current to the same extent as is a heart beating slowly and feebly by a strong current.

The contraction or beat thus produced by a single induction-shock is generally followed by a pause, much more evident in some cases than in others. We have not been able to discover the exact conditions which determine the duration of the pause; but it seems to depend much more on the state of the heart than on the strength of the current. There appears to be no necessary relation between the amount of the contraction and the length of the consequent pause. Thus a contraction twice the strength of the normal beat is not invariably followed by a pause of twice the length of the normal diastole; it may be more than twice as great, it may be hardly greater. On this point, again, we speak with hesitation, from inability to measure accurately the force or extent of the beats.

We have thrown in the single induction-shock at all phases of the cardiac cycle, at the height of systole, immediately before and immediately after systole, in the middle of diastole, &c., but have been unable to detect any marked differences in the result. It can hardly be doubted that, whatever be the way in which the shock causes a contraction, the heart must be differently disposed towards a stimulus, according as it is about to make or has just made a beat; nevertheless we have not succeeded in observing any marked differences.

Not only has the contraction produced by a single induction-shock the general characters of a normal "beat" rather than those of a simple muscular contraction, but, as far as can be judged by the eye, the effect of the shock on the ventricle differs from the effects produced in any ordinary muscular fibre in the following respects. In passing gradually from extremely feeble to stronger currents, there is not witnessed a gradual increase of effect; on the contrary, the change is a sudden one, from apparently no effect at all to the production of a well-marked beat. This leads us to infer that the mechanism by which feeble currents produce contractions in the heart differs somewhat from that whereby contractions are caused by stimulating an ordinary muscular fibre*. The stronger contractions, on the other hand, produced by stronger shocks, seem to share more distinctly in the characters of an ordinary muscular contraction.

* Compare Bowditch, *Arbeiten phys. Anstalt, Leipzig*, vi. Jahrgang (1871), p. 139.

We have said that single induction-shocks, below a certain intensity, produce apparently no effect on the heart.

We have thrown in single shocks, of a strength too slight to cause a beat or contraction, at all phases of the cardiac cycle, without being able to detect any variation in either the character or frequency of the beat. We would not venture to say that absolutely no effect is produced, but the effect is not manifested by any change in the rhythm which can be measured on our recording surface, travelling half an inch a second, or by any change in the beat which can be recognized by the eye.

We have also thrown in single shocks repeated at the same phase for several beats in succession. Thus we have thrown in a single weak shock at the height of systole as well as in the middle of diastole, during several successive beats, without detecting any appreciable effects.

Very marked, however, are the results when the weak single induction-shocks are rapidly repeated, so that two or more shocks fall within each cycle.

The application of the shocks produces no contraction or beat, but is followed by a prolongation of the diastole.

In other words, a single induction-shock, too weak to produce by itself any effect, has, when repeated within the cardiac cycle, a distinct inhibitory action.

The beat which immediately follows the prolonged diastole is a feeble one (as are also the beats which occur when the shocks are repeated many times in succession, so as to cover the periods of more than one prolonged diastole); but the inhibition is followed, as was the case with the interrupted current, by a stage of increased rapidity and vigour of beat—that is to say, the inhibition thus caused has all the characters of a normal inhibition.

Repeated trials have shown us that, having due regard to the irritability of the heart, the more feeble the current the more rapid must be the repetition of shocks, and the greater the number of shocks given in order to produce inhibition.

As an instance of very successful inhibition, we may refer to fig. 1, (p. 342), where two shocks were found sufficient to produce a very marked prolongation of the diastole.

A particularly interesting result is shown in fig. 2. This is the only case in which we succeeded in obtaining inhibition as the result of one single opening shock. The shock, however, was (in relation to the heart's irritability) a comparatively strong one; it was just on the verge of being strong enough to produce a contraction. Our notes record that at the moment of stimulation (the stimulus being thrown in very soon after the normal beat) "a slight quiver, quite distinct from any ordinary contraction, and limited to the neighbourhood of the electrodes, was observed."

It will be noticed that after the first two shocks the heart became habituated to the stimulus, and the last two shocks produced no effect.

The "slight quiver" recorded in the above observations does not really contradict the statement made above concerning the rapid transition from a state of rest to a full though slight beat. The transition from this quiver to a beat is quite as abrupt as that from a state of complete rest.

As far as we have seen, inhibition may be brought about whether the electrodes be placed lengthwise (one at the auricle and the other at the aorta) or sideways, or obliquely.

We have not, however, paid particular attention to the question, whether inhibition can be produced more easily in one direction than another; nor did we, in making the above observations, pay attention to the direction of the current.

It would seem, then, from the facts we have recorded, that the inhibition produced by the tetanizing current, applied directly to the ventricle of the snail's heart, is in reality the simple summation of the effects of the single induction-shocks which make up the tetanizing current.

The effect of each shock is very slight, and a single shock has rarely the power to cause a marked prolongation of the diastole; nevertheless there is an effect which lasts for some time longer than the application of the shock itself, and of such a kind that a second shock being brought to bear on the heart before the effect of the first has passed away, has a cumulative effect, and, in consequence, distinct inhibition is observed, if not always with two, at least with several shocks rapidly repeated.

Moreover this inhibitory effect, this prevention of the contraction or beat, increases with the strength of the current employed up to a certain limit, and then the action of the stimulus is suddenly reversed, and a contraction or beat is caused instead of prevented.

There seems to us to be no escape from this conclusion, that the action of the induction-shock on the tissue of the heart is of such a kind that, beginning with a certain effect (inhibition), as it increases in intensity it suddenly topples over, so to speak, and produces the directly opposite effect (contraction).

We cannot be assisted here by the favourite theory of a double mechanism of inhibition and contraction, with different exhaustibilities, even if we suppose that these mechanisms are evenly distributed all over the ventricle. All exhaustion is, as far as we know, gradual. If we suppose the inhibitory mechanism to be more easily excited and exhausted than the contractile, then in passing from weaker to stronger stimuli we must pass through a phase in which the waning inhibition is just sufficient to counteract the increasing contraction, when, consequently, no effect at all will be produced. In our results, on the contrary, we pass at once from the *maximum* of inhibition to contraction.

The Effects of the Constant Current.

Non-polarizable electrodes (a modification of Donder's pattern) were

always used. The current was supplied by a single Daniell's or Grenet's cell, the circuit being divided between the electrodes placed on the heart and a set of resistance-coils, the resistance in which could be made to vary from .05 ohm to 100,000 ohms, and thus a very great variety obtained in the strength of the current passing through the heart.

It will be as well to begin by describing the effects produced by the constant current on the heart at rest, *i. e.* on hearts which, after removal from the body, did not exhibit any spontaneous beat.

When the ventricle alone was experimented with (the auricle having been removed), and the electrodes placed lengthwise, one against the auricular end, the other against the aortic end of the ventricle, a contraction or beat always took place at the making and at the breaking of the circuit; but the two contractions differed from each other and from a normal beat in a very distinct manner.

The normal beat of the ventricle may be described as a gathering of the whole mass towards the point where the long axis of the ventricle would be intersected by a transverse line drawn across the ventricle's broadest part, accompanied by a kind of peristaltic constriction, which travels down from the auricular to the aortic end, *i. e.* which begins at the auricular and leaves off at the aortic end, though during the greater part of the period of the beat the whole of the ventricle is constricted. These features of the beat may be recognized in an ordinary beat, and become very obvious when the beat is abnormally prolonged, as when under the influence of certain drugs (atropine &c.). From this normal beat the beats or contractions caused by the closing or opening of a constant current differ in the following way.

The closing- or making-beat always starts from the kathode, and travels for a variable distance towards the anode; the opening- or breaking-beat always starts from the anode, and travels towards the kathode, the beat in each being a kind of peristaltic contraction, the features of which are more readily recognized when it starts from the aortic end.

This kathodic making-beat and anodic breaking-beat make their appearance with all intensities of current up to the full strength of a Daniell's cell (beyond this we have never gone), whatever be the position of the electrodes. When the kathode is at the aortic end, the beat begins there; when at the auricular end, the beat begins at the auricular end, and in that case differs chiefly from the normal beat in being more or less confined to that end. When the electrodes are placed at the sides of the ventricle, the kathodic side beats at the making, and the anodic at the breaking, whichever side each be placed.

We have not made many observations on the auricle alone, but have seen enough to satisfy ourselves that the same law holds good there too, that the making-beat is kathodic, and the breaking anodic.

We need hardly point out the remarkable analogy presented by these facts with those resulting from the passage of a constant current through

a nerve, and the support they thus afford to Pflüger's hypothesis, that the establishment of kathelectrotonus and the disappearance of anelectrotonus alone set in motion a stimulus-wave.

When the ventricle and auricle are removed together from the body, and placed between the electrodes, so that one electrode lies at the aortic end of the ventricle and the other at the pulmonary end of the auricle, the ventricle behaves, as far as the make- and break-beats are concerned, very much as if the electrodes were applied directly to the ventricle alone. Thus when the kathode is at the auricle there is a "making"-beat at the auricular end of the ventricle and a "breaking"-beat at the aorta, and *vice versa*.

We have satisfied ourselves by numerous observations that, provided the contraction of the auricle causes no *distension* of the ventricle, the condition of the one chamber of the heart has no effect on that of the other. Between the two is a small ring of a sort of connective tissue, traversed, as far as we can make out, by no nerves, which affords complete physiological isolation of the two chambers. Thus out of the body, empty of blood, auricle and ventricle are two independent organs. Hence when a current is passed in the manner described through both ventricle and auricle, each part undergoes a separate *physiological* polarization, the auricular end of the ventricle becoming cathodic or anodic as the case may be, just as if the current were applied to the ventricle alone, account being taken of the weakening of the current through the additional resistance offered by the auricle. This, at least, is the only explanation we can give of the undoubted fact, that, with some modifications to be mentioned afterwards, the effect of the constant current is the same on the ventricle with the auricle attached as on the ventricle alone.

Besides the mere occurrence of the cathodic making- and the anodic breaking-beat, there are several facts worthy of attention. These we have studied chiefly on the ventricle with the auricle attached; but we see no reason to doubt that the same facts appear when the ventricle alone is used.

When the period during which the current passes is very short, when the current is very rapidly made and broken, the two contractions are replaced by one. This contraction or beat begins at the kathode, *i. e.* is aortic or auricular according to the position of that electrode.

Here, again, is a curious parallel to the view so generally adopted in reference to the behaviour of nerves towards the constant current, *viz.* that the establishment of kathelectrotonus is a stronger stimulus than the disappearance of anelectrotonus.

When exceedingly feeble currents are employed, it frequently happens that a momentary application of the current, a rapid make and break, produces no contraction or beat. Nevertheless such a current, if applied for several seconds, will give, as usual, a cathodic making- and an anodic breaking-beat.

The absence of a beat on a momentary application might be explained by supposing that the effect of the too rapidly succeeding break interferes with the effect of the immediately preceding make. But, on the one hand, we have no positive indication of the possibility of such an interference (and there are reasons for thinking it extremely unlikely), while the normal character of the kathodic beat, when it does occur with a stronger current, distinctly opposes such a view. On the other hand, a distinct interval does exist between the making of the circuit and the commencement of the contraction, an interval easily recognizable by the eye, though, in the absence of any graphic method, we have been unable to measure it. We conclude, therefore, that, in order to produce the maximum effect of even the making kathodic beat, a certain time is required. This time is longer for weak currents than for strong; and currents sufficiently weak may be applied for a short time without producing any beat, the maximum effect not having time to develop itself, though the beat does appear when the action of the same current is prolonged.

Sufficient time being allowed for the production of the beat, the strength of the kathodic making-beat will depend otherwise entirely on the strength of the current (the irritability of the tissues of the ventricle being supposed to be constant).

The strength of the anodic breaking-beat depends, however, in a peculiar way on the duration of the current. Up to a certain point the more prolonged the action of current the stronger is the anodic beat; but a limit is soon reached, after which the strength of the anodic beat is diminished by further prolongation of the action of the current. We have not gone into this point fully, but the result is evidently connected with a so-called "exhaustion" of the tissue.

We have called the contraction which takes place at the make or break "a beat." We think we are justified in giving it this name, though it differs, as we have pointed out, from the normal beat in some particulars. Our strongest reason for doing so is as follows:—

When the ventricle and auricle are placed between the electrodes, so that the kathode is against the auricle and the anode against the aortic end of the ventricle, the kathodic making-beat is *followed by a second, by a third, and by sometimes several beats*, all kathodic in their features, *i. e.* starting from the kathode and travelling towards the anode. In other words, a rhythmic pulsation is set going at the kathodic end.

It is impossible to regard these beats as simple contractions caused by the stimulus-wave, started, like the stimulus-wave in a nerve, by the establishment of kathelectrotonus. They are evidently actual *beats*, whose occurrence is favoured by the conditions brought about by the action of the current (fig. 10, p. 343.).

Of these the first (the ordinary making-beat) is always (as far as we have seen) the strongest. The succeeding ones diminish in intensity,

and the intervals between the beats generally become increasingly prolonged (see fig. 13).

When the current is maintained for a short time only (10 seconds), the appearance is produced as if the heart were beating during the whole time of the passage of the current (fig. 11).

The anodic breaking-beat is, as far as our observations go, always single, is never followed by a series of beats (that is, of course, when the ventricle is one which is incapable of spontaneous pulsation).

We conclude that the constant current throws the tissue in the neighbourhood of the kathode into such a condition as is favourable for the development of beats; that this effect, though taking some time for its production, reaches its maximum very soon after the making of the circuit, and thenceforward diminishes gradually.

In the neighbourhood of the anode, on the other hand, the tissue is thrown into a condition unfavourable for the production of beats, the beats which have originated in the kathodic region ceasing as they approach the anode. The rebound, however, which follows upon the breaking of the circuit develops a beat, with the occurrence of which the tissue returns to a normal condition of equilibrium, and no further beats occur.

Such are the facts which may be observed when the kathode is placed against the auricle, and the anode against the aortic end of the ventricle.

One would naturally expect that similar results, *mutatis mutandis*, would be obtained when the kathode was placed at the aortic end of the ventricle, and the anode at the auricle. Such, however, is not the case.

When the kathode is at the aorta there is only a kathodic making- and an anodic breaking-beat. Between the two the ventricle, as a general rule, remains perfectly quiet (see figs. 12 & 9). There is therefore a functional difference between the auricular and aortic ends of the ventricle.

Led by the apparent analogy of the vertebrate heart, one might be inclined to infer that there was an automatic mechanism present at the auricular end of the ventricle, but none at the aortic end.

This view, however, is directly negatived by the following facts:—

The aortic end will, in active and favourable hearts, continue to beat spontaneously for some time after being separated from the rest of the ventricle.

When, as we shall presently have occasion to mention, a spontaneously beating ventricle is submitted to the action of the constant current, the beats begin at the aortic end, and are frequently entirely confined to the aortic end when that end is made kathodic. This would be impossible if the automatic mechanism were confined to the auricular end. We have been led to connect the explanation with the shape of the ventricle

The greater mass of the contractile tissue of the ventricle lies towards the auricular end. If the ventricle were supposed to be bisected transversely by a straight line drawn across it, and its constituent fibres regarded as so many straight lines, representing so many forces, those forces might be considered as applied at a point on the auricular side of that straight line.

If we, then, suppose (we shall presently state our reasons for such a supposition) that the rhythmic impulse is generated by changes in all the fibres, that impulse would naturally manifest itself first, as in fact it does, at the auricular end.

Moreover, since the heart was first formed, each beat has been taking place after a fixed normal fashion, beginning at the auricular end. Hence the nutrition, the life (let it be called what it may) of each part is habituated and regulated to such conditions as are involved in the normal beat beginning at the auricular end. One would therefore expect that, even under abnormal conditions, it would be easier to call forth a beat beginning at the auricular end than at the aortic end, and that when the heart is divided into two pieces the auricular end would manifest a rhythmic beat more readily than the aortic end, though each end were absolutely possessed of the power of rhythmic pulsation.

This is actually the case. The aortic end, separated from the auricular end, will, under favourable circumstances, beat spontaneously, but will not do so with the same readiness as will the auricular end. Under the influence of the constant current, the beats of a spontaneously beating heart may be made to begin at and be confined to either the aortic or the auricular end, but not indifferently; they appear most readily at the auricular end. Under the influence of the constant current, the heart, otherwise at rest, may be made to beat spontaneously at the auricular end, but not at all, or with great difficulty, at the aortic end.

So far concerning the action of the constant current on the ventricle at rest.

We have already been led to anticipate some of the results gained by applying the constant current to a heart spontaneously beating.

These are somewhat more difficult to analyze, on account of the greater susceptibility of the tissue, and the greater variations in irritability resulting from treatment.

Thus, when the current used is one so strong that the momentary application of it is followed by a powerful contraction, it is evident that in dealing with the effects of the same current applied for several seconds, we have to consider that the powerful initial "making" contraction must influence, by the reactions following it, the behaviour of the heart during the passage of the current. In the same way the breaking of the current will be followed by events the causes of which are sought for partly in the reaction following upon the strong contraction caused by the break.

To avoid these difficulties we have generally employed a current so weak, that a momentary application of it to the beating heart produced no appreciable result.

During the passage, however, of such a weak current very marked effects make their appearance.

Whatever the position of the electrodes, whether the current be directed longitudinally, with the kathode at the auricular end or at the aortic end, or transversely or obliquely, in all cases the normal beat is modified in such a way that the contraction begins at the kathode, and is more or less limited to the neighbourhood of the kathode. Thus, when the kathode is placed at the auricular end of the ventricle, the part immediately round the kathode contracts first, and the contraction passes down in a vermicular manner towards the anode, sometimes a very little way, sometimes a considerable distance. When the kathode is placed at the commencement of the aorta, the contractions begin at the aorta and pass upwards towards the auricular end.

Being thus partial, the beats may be said to be weaker than normal, the more so the more they are confined to the kathode. Their feebleness seems to be more evident with stronger currents (not so strong, however, as to produce an initial contraction) than with weaker ones.

We have not been able to satisfy ourselves as to any marked or constant change in the rhythm taking place.

These kathodic beats are succeeded, on breaking the current, by one or more markedly strong beats of a reverse direction, beginning at the anode and travelling towards the kathode.

In some cases the reversal has been seen to take place during the passage of the current, the last few beats beginning at the anode, but not then exhibiting the same increase of strength which is seen in the anodic beats succeeding the breaking of the current.

This last observation illustrates the fact that the effect of the constant current, like that of the tetanizing current, is at its maximum effect near the commencement, and thence declines with greater or less rapidity, the tissue becoming insensible to the current. In this case insensibility was reached after the current had been passing a very short time; and it is interesting to remark that, though the direct effect of the current thus became lost, the reaction caused by the action of the current in the first part of the period was able to manifest itself by the anodic beats.

Thus, like the initial and final beats observed when the ventricle is at rest before the application of the currents, the apparently spontaneous beats always originate at the kathode, are more or less confined to the kathodic region, and, according to the position of the electrode, may be witnessed either at the auricular or at the aortic end.

As with the heart at rest, however, so with the heart in spontaneous movement, there is a functional difference between the aortic and the auricular ends.

This difference is most readily observed when the ventricle with the auricle attached is placed between the electrodes than when the ventricle alone is subjected to the action of the current, though we have observed the same phenomena in the latter case also.

When the anode is placed at the aorta, and the kathode at the auricle, a very feeble current produces no appreciable effect, except that perhaps the beats are rather feebler and somewhat quicker (fig. 5, p. 342).

The same current, applied to the same heart in the opposite direction (that is, with the kathode at the aorta and the anode at the auricle), *distinctly inhibits the ventricle*, so that it remains in diastole during the whole time of the passage of the current, and resumes its beat on the current being broken.

This inhibition is very clearly shown in fig. 7. Here advantage was taken of a curious secondary rhythm (fig. 6), which we have had more than one opportunity of witnessing. The ventricle, after a period of quiescence, began to beat, at first feebly with long pauses, then more rapidly and strongly. Having reached a maximum, the beats similarly declined, and thus a new period of quiescence was again begun. This alternation of rhythmic beats and quiescence was observed for a long time.

It will be seen that when the constant current (which was exceedingly weak, inasmuch as the resistance-circuit only offered a single ohm, or even only a fraction of an ohm resistance) was thrown into the heart at one of the ventricle's beating periods, no effect was produced when the anode was at the aorta, but distinct inhibition when the kathode was at the aorta.

During the period of quiescence the effect of the current is very much what we have already described as that of the constant current on a ventricle which has ceased beating, viz. that when the anode is at the aorta, and when, consequently, the auricular end of the ventricle becomes kathodic, a rhythmic beat tends to make its appearance, but when the auricular end is made anodic, no beats make their appearance (fig. 8).

The explanation we would give of these facts (very similar to that given at p. 328) is as follows:—

The current employed was a very feeble one, rendered all the more feeble, as far as its effect on the ventricle was concerned, by the fact of some of it having been occupied in effecting the polarization of the auricle.

When the kathode is placed at the aorta, the auricular end of the ventricle becomes anodic. Hence, as we have already seen, the beats would, if the current were of a sufficient intensity, while absent at the auricular end, be present at and start from the aortic end. We have already seen that the auricular end is more sensitive than the aortic end. Hence, with a sufficiently weak current, as in the case in point, the auricular end being more affected than the aortic end, the beats are inhibited at the auricular

end while as yet the current is unable to produce the beats at the aortic end, and the result is an apparent general inhibition of the whole ventricle.

The inhibition thus produced is, as far as we can see, a distinct inhibition; that is, the heart, which previously was beating regularly, stops beating when the current is thrown into it, remains in diastole during the short time the current continues to pass through it, and resumes its beat on the current being removed.

The occurrence of inhibition is, then, here a matter of degree; it depends on the existence of a certain relation between the irritability of the ventricle and the strength of the current employed. When a stronger current is employed, the beats, while continuing to be absent at the anode, make their appearance at the kathode, and the heart, though beating quite differently from the normal, can no longer be said to be inhibited. There is no room here for any theory of a special inhibitory mechanism, except such a one as would suppose that while the automatic mechanism was exalted at the kathode and depressed at the anode, the inhibitory mechanism was by weak currents exalted at the anode and depressed at the kathode. Such a view would be either simply a clumsy expression of facts or, if any thing more, directly opposed to all our experience of the behaviour of irritable living matter towards electric currents.

We would now call attention to fig. 5. This is the same heart which was inhibited by a current passing from the auricle to the aorta, and is apparently but little affected by nearly the same strength of current passing from the aorta to the auricle. But it will be noticed that, though the throwing of the auricular end of the ventricle into a kathodic condition does not very much, if at all, increase the beat of the ventricle, the withdrawal of the current is followed by a very distinct pause (*a*)—in fact, by an inhibition of short duration.

This pause must be due to a reaction taking place in the kathodic region. It can hardly be due to a reaction taking place at the anodic or aortic region; for, as we have already seen, reaction at the anode takes on the form of a beat or contraction. We thus get this remarkable result, that at the kathode, where the action of the current during its passage is favourable to the beat, *the after effects, which are in the way of inhibition, are more marked than the effects of the current itself, which, as far as they go, are in the way of a quickening of the beat.*

In other words, at the auricular end of the ventricle, of whose condition, by reason of its greater susceptibility, we are better able to judge than of that of the aortic end, we find that, whatever be the direction of the current, the total effect of the passage of the current is inhibitory. For when the auricular end is anodic, the current produces on that part of the ventricle a direct inhibitory effect, which the exalted (kathodic) condition of the aortic end is unable to counterbalance; and when the auricular end is kathodic, the depressed (anodic) condition of the aortic

end hinders any great increase in the total force of the beats—so that the direct effect of the current is manifested only, if at all, in a quickening of the beat, while the after effect is distinctly inhibitory, to such an extent as not to be obscured by the reaction, in the way of exaltation, which takes place at the (anodic) aortic end. In other words, whichever its direction, in the one case by action, in the other by reaction, a sufficiently weak current leaves a *balance* in favour of inhibition.

Though, by reason of the less susceptibility of the aortic end and the consequently slighter development in that region of a rhythmic beat, we are less able to judge of what is going on in that region during the passage of the current, we may, we venture to think, assume that the changes there are of fundamentally the same kind as at the auricular end—that is to say, that the total effect of the passage of the current would at this end too be greater *in the direction of inhibition* than of exaltation. A sufficiently weak current, then, passed lengthwise through the ventricle in either direction, whether from aorta to auricle or from auricle to aorta, has an inhibitory effect (when the period of reaction is included) greater than its exalting effect.

If we suppose such a current to be momentarily passed through the ventricle between any two beats, its effect therefore, as far as it went, would be to prolong the diastole. The effect might not be very obvious; it might be so small as to escape detection by itself, but it might be made manifest in the following way:—The effect of the current, as we have in all cases seen, whether exalting or inhibitory, takes some time to establish, and lasts longer than the actual passage of the current. Hence, if a momentary current like the above be succeeded before the next beat by a second similar one, the effect of the second would be added to the first, and the diastole still further prolonged. By means of momentary currents, repeated so rapidly that the effect of one had not passed off before the next began to tell, the diastole would be so prolonged that the inhibitory effect of the current would be undeniable.

Moreover, though we have used the longitudinal application of the current as a means of analyzing its effects, there is nothing to prevent a similar explanation being given of the inhibitory effects of a weak momentary current passed transversely, or in any direction, through the ventricle or through the auricle. The inhibitory effect of the longitudinal current depends essentially on the fact that the total effect of the current, when the after effects are included, is one of depression, though it has partial elements of exaltation; and this will hold good whatever be the position of the electrodes, provided that the current be of a certain intensity in relation to the irritability of the tissue.

We conclude, then, that the clear and obvious inhibition brought about by sufficiently rapid repetitions of momentary currents (such as single induction-shocks) of a certain feebleness is due to the summation of a number of slight inhibitions, the occurrence of each single inhibition

depending on the fact that the total depressing effect of the current on the rhythmically beating tissue is greater than the total exalting effect.

It may seem strange to speak of a stimulus as producing a depressing effect; but reflection will show that it is quite what ought to be expected.

We have in the foregoing contented ourselves with speaking of the tissue in the neighbourhood of the electrodes as being in a kathodic or anodic condition as the case may be. We did so because, not having examined the condition of the electric currents of the ventricle, we hesitated in using the terms kathelectrotonus and anelectrotonus.

We know, however, that muscle is capable of being thrown, *in the intrapolar region*, into an electrotonic condition, and that (quite in accordance with all the results recorded above) this condition lasts after the removal of the current. We therefore shall probably not err in supposing that the polarization of the cardiac tissue, which is obviously a result of the constant current, is more or less allied to that kind of polarization which we call electrotonus.

Now the electrotonic condition of nerve or muscle differs essentially from the polarization of any dead matter in this, that it is *essentially a function of the vital activity* of the substance polarized. May we interpret this as meaning that the change in muscle or nerve which constitutes the electrotonic condition is brought about and maintained, not solely at the expense of the energy of the current, but also, perhaps chiefly, *at the expense of the energy of the tissue itself*?

If so, then we may argue that while in the normally beating heart all the energy of the tissue is used for the production of the beat, when a constant current is passed through it some of this energy, in addition to the energy of the current itself, is used in establishing anelectrotonus and kathelectrotonus. There must therefore, as the result of the polarization, be less energy (for the time being) available for the purposes of pulsation.

The idea of the exalting influence of a stimulus arises naturally from the fact that the stimulus may, if sufficiently intense, give rise to a contraction; and it seems at first sight contradictory that one and the same thing should produce apparently exactly opposite effects.

It is very easy, however, to trace, in the beating ventricle of the snail's heart, the transition from inhibition to the production of a contraction or artificial beat.

One stage in this transition is shown in fig. 4.

Here a weak constant current passed through the ventricle longitudinally from auricle to ventricle (and thus most favourable for inhibition) caused, when the passage was momentary (*i. e.* was removed before it had time to produce its full effect, and thus was reduced, so to speak,

in its action much below its normal strength), simply a brief inhibition, indicated by the greater length of the diastole marked *a*.

The same current, allowed to act on the tissue a sufficient time for it to produce its maximum effect, gave rise to a making- and breaking-beat, the making-beat being what we have called kathodic (starting from the aortic end in this case), the breaking anodic (starting from the auricular end). The current was sufficiently strong while inhibiting the auricular end to cause a kathodic beat of the aortic end.

This same current would, if sufficiently increased in strength, have been able to produce, on a momentary passage, the same effect as it has in the above observation by prolonged passage, with this exception, that there would be one visible beat only instead of two.

In this way, by simply varying the intensity of the current, we get either inhibition or contraction.

(It will be observed that in fig. 2 the kathodic beat is followed by a pause, as is also the anodic beat; and a single contraction produced by the momentary application of a stronger current would show a similar pause. These pauses may be regarded as partly due to the effects of the contractions—that is, to what is commonly spoken of as exhaustion; but it is obvious, from all our preceding observations, that they are partly due to the direct inhibitory effect of the current.)

There is, then, no absolute contradiction in the fact of a current producing either inhibition or a beat, according to its intensity; and even when a beat does take place, the essentially depressing effect of the current is seen in the subsequent pause or inhibition, generally spoken of as exhaustion.

With the occurrence of a contraction, however, the direct effects of the current become mixed with the effects and after effects of the changes resulting in the shortening of the fibres; and thus the pause after a contraction has laws of its own, more or less different from those of the simple inhibitory pause, and is more difficult to analyze.

On a Secondary Rhythm.

We would wish to call attention to the peculiar secondary rhythm, an example of which is shown in fig. 6. The characters of the rhythm are very marked. The heart, after remaining for some seconds in an apparently perfectly quiescent state, gives a beat hardly recognizable even under the microscope. This is succeeded by feeble beats, which, though not with complete regularity, increase in force, with shortening intervals, until a maximum is reached, after which the beats decrease in force, and the intervals lengthen, until perfect quiescence is again reached.

A somewhat similar secondary rhythm has been observed in the vertebrate heart as the result of prolonged stimulation of the pneumogastric nerve (compare Luciani, *Arbeiten phys. Anstalt, Leipzig, 1873, vii. Jahrgang, p. 113*). We have noticed it several times in the snail's heart;

and, as far as we know, the heart had in each instance been treated, in some way or other, by electric currents.

We are totally unable to give any explanation of the phenomenon; and though we have tried in various ways to produce it artificially at will, have always failed to do so.

It suggests the curious inquiry whether, during the quiescent periods, the heart is absolutely at rest, or in reality executing pulsations too small to be visible. We have been unable to detect any indications of such invisible beats, and the marked length of the first and last diastole in each active phase would seem to indicate that the median portion at least of each quiescent phase is occupied by a prolonged diastole of absolute quiescence.

We might further speculate as to whether similar secondary rhythms, marked, not of course by absolute quiescence, but by a more or less pronounced rise and fall of cardiac activity, may not exist under normal circumstances—whether they may not, for instance, be diurnal phases of the heart's own nutrition; and also as to whether the normal pulse-phase may not be made up of small, more rapid, oscillations, bearing the same relation to it as it does to the larger rhythm we are speaking of.

Lastly, if the heart of the snail were a barrel-shaped organ, like that of a Tunicate, with each end equipollent to the other, instead of being a conical mass as it is, with a wholly preponderating auricular end, it is easy to see how such a secondary rhythm as the one we have described would, if started at each end of the tube at different times, produce the well-known alternating action so characteristic of the Tunicate heart.

Experiments on the Hearts of other Mollusks.

The foregoing observations on the heart of the snail were made by us together in the Physiological Laboratory at Cambridge.

During a stay at the Zoological Station at Naples, in the month of November last, Mr. Dew-Smith made several observations on the hearts of *Sepia* and *Aplysia*, with the view to ascertain whether they would behave towards electric currents in the same way as the heart of the snail. We hoped, too, they perhaps might be found more convenient for the purposes of experiment; this hope, however, was not realized.

In the first place the heart of the *Sepia*, though it will beat when removed from the body, can be got to do so with extreme difficulty, and then for a short time only. The observations consequently had to be conducted on the heart *in situ*, and for this reason, as was explained in the case of the snail, were robbed of much of their value.

Moreover the peculiar shape of the ventricle of the *Sepia's* heart, with its two aortæ and its two branchial sinuses, render it much less suitable for the purpose in hand than the more compact and simple ventricle of the snail.

Nevertheless the following results were arrived at:—

Cut into two, so as to divide one aorta and the branchial sinus of one side from the other aorta and the branchial sinus of the other side, each half of the *Sepia's* heart continued to beat rhythmically.

Hence there cannot be a single automatic nervous centre for the ventricle of the *Sepia's* heart.

No nerve could be discovered, the stimulation of which would produce inhibition of the heart after the fashion of the vertebrate pneumogastric.

The interrupted current applied directly to the ventricle did, under favourable circumstances, produce a stoppage of the rhythm, the heart remaining in diastole and resuming its beat on the removal of the current.

Single induction-shocks and the make and break of a constant current appeared to produce the same effects in *Sepia* as in the snail, sometimes causing a contraction and sometimes having as a result a more or less distinct inhibition.

During the passage of a constant current exactly the same phenomena were apparent as in the case of the snail, the beats always starting from the kathode and travelling towards the anode. This effect could, in fact, be seen even more distinctly in the case of *Sepia* than of the snail. By placing the electrodes in a proper position the course of the pulsation along the ventricle might be at pleasure reversed.

In general we may, we believe, safely conclude that what we have found to be the case with the snail's heart, holds good also, in fundamental points, for the heart of *Sepia*.

It was hoped that the large specimens of *Aplysia* which were at hand at the Station would prove of valuable assistance. The difficulty of working with them, owing to the contractions of the body and the unwillingness of the heart to beat out of the body, prevented, however, any satisfactory observations being made with them.

As in *Sepia*, so in *Aplysia*, no nerve could be found, stimulation of which would cause direct inhibition of the heart.

Three specimens of *Salpa* came into Mr. Dew-Smith's hands.

In these two platinum electrodes were thrust through the body, so that their points came to the neighbourhood of the heart, the pulsations of which could be readily observed with the naked eye and counted. The position of the electrodes was such that a current sent through them would pass in large measure lengthwise through the heart.

The effect of the interrupted current was very remarkable. During its passage the heart, which, according to wont, had previously been alternating in direction after every 6 to 15 beats, continued to beat for two minutes *entirely in one* direction at a quickened rate. No inhibition could be produced, even with strong currents.

We quote this single observation because it was the only one that could be made. Bad weather setting in prevented any more *Salpa* being obtained before Mr. Dew-Smith left for England; otherwise it would

have been extremely interesting to have studied the action of the constant current.

General Considerations.

The beat of the ventricle of the snail's heart may, we venture to think, be regarded as a rhythmic movement of purely protoplasmic nature.

The constituent fibres, if fibres we may venture to call them, are not isolated like the fibres of a vertebrate voluntary muscle, but physiologically continuous; so that any change set up in any part of the ventricle can be propagated over the whole of it in the same way that a contraction-wave set going at any point in a striated fibre is propagated along the whole length of the fibre, or that a contraction-wave is propagated from the point of stimulation along a nerveless ureter. (Compare Engelmann, Pflüger's Archiv, ii. p. 243.)

[The ease with which the entire ventricle of the snail can be completely polarized by the constant current may be regarded as another proof of the physiological continuity of its tissue. The want of conformity between the directions of its fibres and those of the paths naturally taken by the current in passing from one electrode to the other, forbids us to suppose that the effects described above can be the combined result of a number of independently polarized fibres.]

The changes which result in the rhythmic beat take place normally in all parts of the tissue, so that any moiety of the ventricle isolated from the rest is, under satisfactory nutritive conditions, able to execute rhythmic movements. (The isolation may be mechanical, as by section, or physical, as by polarization with the constant current.)

When the ventricle is cut in half, the two halves do not necessarily beat synchronously. Each half has a rhythm of its own, which may or may not be (and in nearly every instance is not) the same in both halves. But the rhythm of each half is, under favourable circumstances, perfect and complete; and the same may be said of still smaller pieces.

Now the normal beat of the entire ventricle is a complicated act. There are in it definite sequences. Certain fibres or certain parts of the tissue begin to contract before others, and certain parts continue to contract after others have ceased to do so. The beat is not a simple contraction-wave passing uniformly from one end to the other, or radiating equally in all directions from one point, but a peculiar movement, having for its object the ejection of fluid from the cavity in the best possible manner, and is hence a coordinated movement.

The aortic half of the ventricle, separated from the auricular half, starts each of its beats quite independently of what is going on in the ventricular half, and *vice versa*. When the two are physiologically continuous, the changes in the one are determined by the changes in the other. In order that a normal beat may be fairly carried through, the auricular half must not start its contraction until the aortic half is ready to

carry it on. Otherwise there would be not a regular beat, but an irregular skirmish of contractions.

We are driven, therefore, to conclude that in the normal condition, where the tissue is physiologically continuous, there are means of communication between all parts; so that the auricular half, for instance, on the one hand, feels, if we may use the phrase, what is going on in the aortic half, and, on the other, exerts an influence on those changes in order to accommodate them to the changes taking place in itself, and *vice versâ*.

We believe that all our knowledge of protoplasm (and we might point especially to the harmonious working of groups of cilia and ciliated cells) favours the idea of the existence of such a *consensus* among the several parts of any mass of protoplasm which acts together as a whole. Nor ought there really to be any difficulty in supposing such communications to be effected by molecular movements in the undifferentiated protoplasm; for there must be in protoplasm many kinds of currents and internal motions for which we have at present no names. We might further urge that there must be in undifferentiated protoplasm the *rudiments* of all the fundamental functions present in the differentiated structures of higher animals. Thus the process by which the condition of the aortic region of the snail's ventricle is communicated to the auricular region seems to be the rudiment of the *muscular sense*.

Adopting some such view as this, we are naturally led to what we venture to think is a more satisfactory interpretation of the part played by ganglia in rhythmic and automatic movements than the one commonly adopted.

The prevalent conception, based on the view put forward by Sir James Paget in his well-known Croonian lecture, if we understand it aright, is somewhat as follows:—Taking, for instance, the automatic ganglia of the heart, motor impulses are rhythmically generated in the ganglionic nerve-cells, as the result of the nutrition of these nerve-cells themselves, influenced, according to circumstances, by afferent impulses starting from sentient surfaces internal or external of the heart itself, or brought from afar by the pneumogastric or other nerves.

These motor impulses reaching the muscular fibres call forth a beat, the contractile elements being, so to speak, mere passive instruments of the nerve-cells, their condition affecting the beat only so far as its force is concerned, inasmuch as, with the same stimulus, the contraction will be greater in a more irritable, and smaller in a less irritable fibre.

Now every one must have felt a difficulty in supposing that the nutrition of a few minute nerve-cells should be all in all, while that of the large mass of the continually and rapidly changing muscular tissue should go for nothing in determining what is at least quite as important as the force of each contraction, viz. the rate of rhythm. Moreover such a view puts, to a certain extent, asunder what nature has evidently, by intricate

ties, joined together—the force and character of the systole and the length of the diastole.

We would suggest that the ganglia are not automatic in function in the above sense, but simple coordinators. The contractile tissue of the frog's ventricle, arranged in bundles isolated by connective tissue, is not physiologically continuous, though each bundle formed by the opposition of branched sheathless muscular fibres may be continuous throughout itself; and the consensus we spoke of just now cannot be effected by molecular communications. Hence the existence of differentiated organs, in the form of nerves and ganglia, by means of which indications of the condition of the isolated constituents of the ventricle (and, we may add, of the isolated auricles and ventricle) are carried, *as items of a muscular sense*, to a central organ, and thus the state of each part is made common to all. In this central organ the advent and character of each beat is determined as the expression of the nutritive condition, not of the nerve-cells only, but of the contractile elements as well.

If this view of the function of automatic ganglia (which, it will be observed, is simply a modification of Sir J. Paget's conception) be correct, it is easy to understand why we have found no such organs in the physiologically continuous protoplasmic heart of the snail.

It needs no such ganglia for the carrying on of its own rhythmic beat; nor does it need them to place it *en rapport* with the rest of the body of the animal. The movements of the body determine the quantity of blood flowing to the heart; the force and rapidity of the heart's beat is in direct ratio to the quantity of blood distending its cavities; and thus a harmony is established between the movements of the body and the circulation quite sufficient for the purposes of the snail's life, without the intervention of that nervous regulative mechanism supplied to the vertebrate heart by the various cardiac ganglia, the pneumogastric, and other nerves.

Résumé.

We have seen that, in the region of the kathode, a condition which we have compared with the kathelectrotonus of nerves is set up. This condition we will not at present venture to characterize more closely than to say that it is favourable to the production of rhythmic beats. At the anode precisely the opposite effect takes place.

We have shown that both conditions require some time for their complete establishment, and that when established they at once begin to decline. Thus they speedily reach a maximum and more gradually subside.

The setting up of kathelectrotonus will, if sufficiently intense and the maximum be fairly reached, give rise to a contraction or beat. The giving way of anelectrotonus, though with less ease, will also cause a beat. Both the establishment of anelectrotonus and the disappearance of kathelectrotonus are unfavourable to the production of the rhythmic beat.

Thus the effect produced by a constant current at each pole is mixed, being partly favourable and partly unfavourable to a beat.

The total effect of the passage of a current, whether a contraction be called forth or not, is unfavourable to the rhythmic beat; and when currents too weak or of too short duration to produce a visible contraction are employed, the unfavourable effect alone becomes evident.

When the brief passage of a weak current is followed at a sufficiently short interval by a similar brief passage, the unfavourable effects are, in a measure, added together; and thus, by repetition, a cumulative result is reached.

Hence it is that a single induction-shock, too weak in itself to produce either a contraction or such a prolongation of the diastole as can be satisfactorily measured, will, if repeated rapidly enough, produce an unmistakable inhibition.

How far the observations we have made, and the explanations we have offered, can be regarded as throwing any light on the working of the vertebrate heart and on the general theory of inhibition, must be determined by further inquiries. These we have already commenced, and hope before long to lay some of our results before the Society.

EXPLANATION OF THE DIAGRAMS (pp. 342, 343).

They are all to be read from right to left.

Each figure consists (except in the case of fig. 6) of three lines. Of these, one, generally the upper, indicates the application of the stimulus.

In the case of single induction-shocks, as in figs. 1 & 2, the descent of the line indicates the making, the ascent the breaking of the primary current. Inasmuch as the making-shock was alone employed, the breaking-shock being stopped, the descent in these figures alone indicates stimulation.

In figs. 3-13 the ascent in the line, generally indicated by *x*, marks the making, and the descent, generally marked *y*, indicates the breaking of the current.

The middle line (unless otherwise mentioned) indicates the beat of the ventricle.

In figs. 1-8 the ascent, and in figs. 9-13 the descent, marks the commencement of the beat.

In no case does the interval between the ascent and descent in figs. 1-8, or descent and ascent in figs. 9-13, in any way correspond to the length of the ventricular systole.

The marks on the bottom line in each figure indicate seconds.

Fig. 1. Repeated single induction-shocks.

Two shocks thrown in at *x* causing distinct prolongation of diastole *a*, then followed a very feeble beat, then two more shocks at *y*, followed again by prolongation of diastole *β*. The stimulations were produced by a Du Bois-Reymond's induction-apparatus, worked by one Daniell's cell. The secondary coil at 8°. The making-shock only was used.

Fig. 2. Single induction-shock.

Single induction-shock thrown in at *x*, after a series of normal beats. The result was distinct inhibition for a time, then followed a feeble beat at *a*, then another shock was thrown in at *y*, followed by a pause. At *β* the heart commenced to beat again, and resisted the influence of the three succeeding shocks thrown in, continuing to beat normally. Making-shock only. One Daniell's cell. Coil at 5°.

Fig. 3. Constant current applied to a spontaneously beating ventricle.

The upper line indicates the beats of the ventricle, the middle line the application of the current. The kathode was placed at the aortic end of the ventricle, anode placed at the auricular end of the ventricle. No resistance in the resistance-shunt; consequently only the very smallest fraction of the current could have passed through the ventricle. Current made at x and broken at y .

The beat α started from the aortic end, the beat β from the auricular end.

Fig. 4. The same current, applied to the same heart, under exactly similar circumstances as in fig. 3, except that the break almost instantaneously followed the make. No beat was produced, but only a lengthening of the diastole α .

Fig. 5. Effect of constant current applied to the spontaneously beating heart, the auricle still attached to ventricle. Kathode at auricle, anode at aortic end of ventricle. Resistance introduced into the resistance-shunt *nil*; consequently the very feeblest current was sent through the heart. The current is made at x and broken at y . No appreciable effect during the passage of the current, except a slight quickening of the beat. The breaking of the current is marked by the prolonged diastole at α .

Fig. 6. Example of the "secondary rhythm" of the ventricle of the snail's heart.

Shows the secondary rhythm spoken of on page 334. The last of a series of 9 beats is shown at α . Then follows a pause (β) for 18 seconds; then the beats recommence at γ , going on 9 times to 8; then again follows the pause ϵ , succeeded by another series of 9 beats, commencing at ζ .

Fig. 7. Inhibition of ventricle by constant current thrown in during an active period of the secondary rhythm shown in fig. 6.

Auricle still attached to ventricle, and .1 ohm introduced into the resistance-shunt. The anode at the auricle, the kathode at the aortic end of the ventricle. An instantaneous make and break, whether thrown in during the period of rest or during the period of activity, produced no effect. α is the first of the series of beats succeeding a pause. On the making of the current at x the beats cease, but reappear at β upon the breaking of the current at y .

N.B. The point of the lever marking the ventricular beats on the upper line was not placed exactly over the point of the lever marking on the middle line the application of the current, consequently the beat β seems to precede the breaking of the current at y . In reality it succeeded it by a very short interval.

Fig. 8. Pulsation brought on by constant current thrown in during the resting-period of secondary rhythm.

Heart and strength of current same as in fig. 7. Kathode at auricle, anode at aortic end of ventricle. The resting-period or pause is seen commencing at α , but is broken by the beat β , induced by the constant current, which is made at x ; the beats continue, ceasing at γ when the current is broken at y .

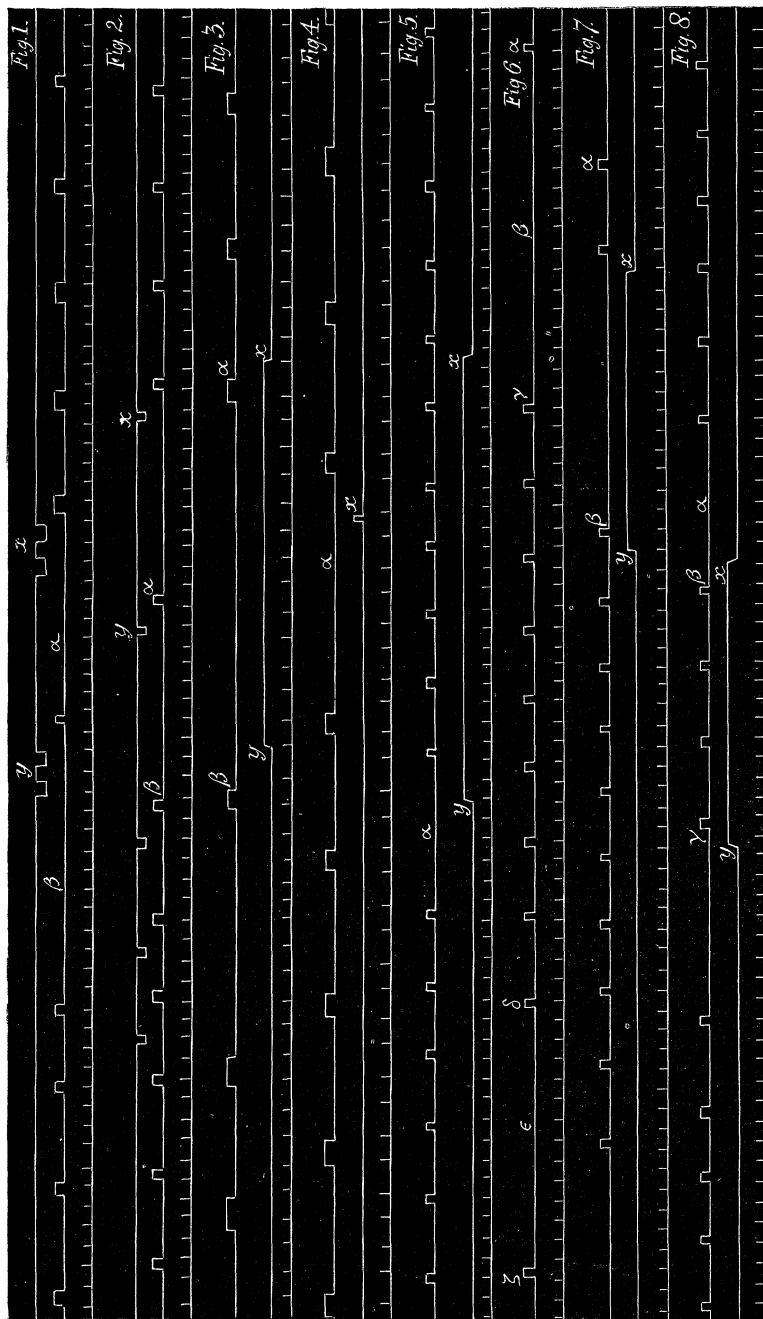
Fig. 9. Constant current applied to the heart at rest.

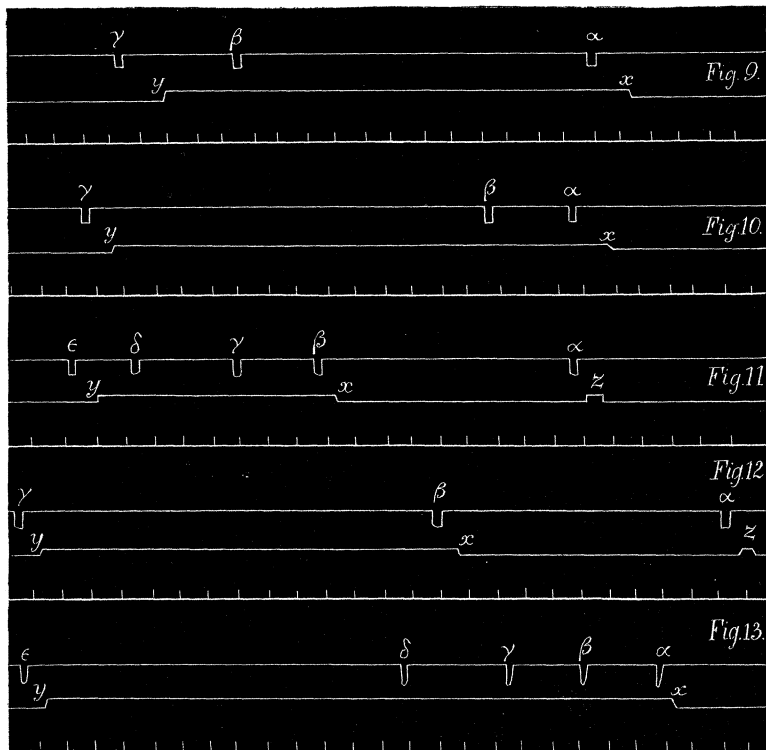
Auricle still attached to the ventricle. Anode at the auricle, kathode at the aortic end of the ventricle. The resistance in the resistance-shunt amounted to 1 ohm. Otherwise the arrangements were the same as in fig. 12 &c. The beat α was very faint, starting from the aortic end of the ventricle. The beats β , γ started from the auricular end of the ventricle. This was an exceptional case, in which a beat (β) appeared before the breaking of the current.

Fig. 10. Constant current.

Applied to heart at rest. Kathode at auricle, anode at aortic end of ventricle.

The top line indicates beat of ventricle; the middle line indicates the application of the current, made at x and broken at y . A single Daniell's cell was used. In the resistance-shunt the resistance was 1 ohm; consequently a small fraction only of the total current passed through the heart.





The beats α, β commenced at the auricular end of the ventricle; the beat γ , due to disappearance of anelectrotonus, commenced at the aortic end of the ventricle.

Fig. 11. Same arrangement as fig. 10. Resistance in the resistance-shunt 100 ohms.

The beat at α , due to the instantaneous make and break at z , was normal in character, *i. e.* started from the auricular end of the ventricle. The beats γ, δ also started from the auricular end. The beat ϵ started from the aortic end of the ventricle.

Fig. 12. Constant current applied to the heart at rest.

Auricle still attached to ventricle. Anode placed at auricle, kathode at aortic end of ventricle. The whole of the current from a single Daniell's cell was sent through the heart. Otherwise the same arrangement as in figs. 10, 12, & 13.

The beat α , due to the instantaneous make and break at z , was faint, starting from the aortic end of the ventricle. β was a stronger beat, starting also from the aortic end of the ventricle. y was a strong beat, starting from the auricular end of the ventricle.

Fig. 13. Same arrangement as figs. 10 & 11.

The whole of the current from a single Daniell's cell was thrown in. The beats $\alpha, \beta, \gamma, \delta$ started from the auricular end, and ϵ from the aortic end of the ventricle.

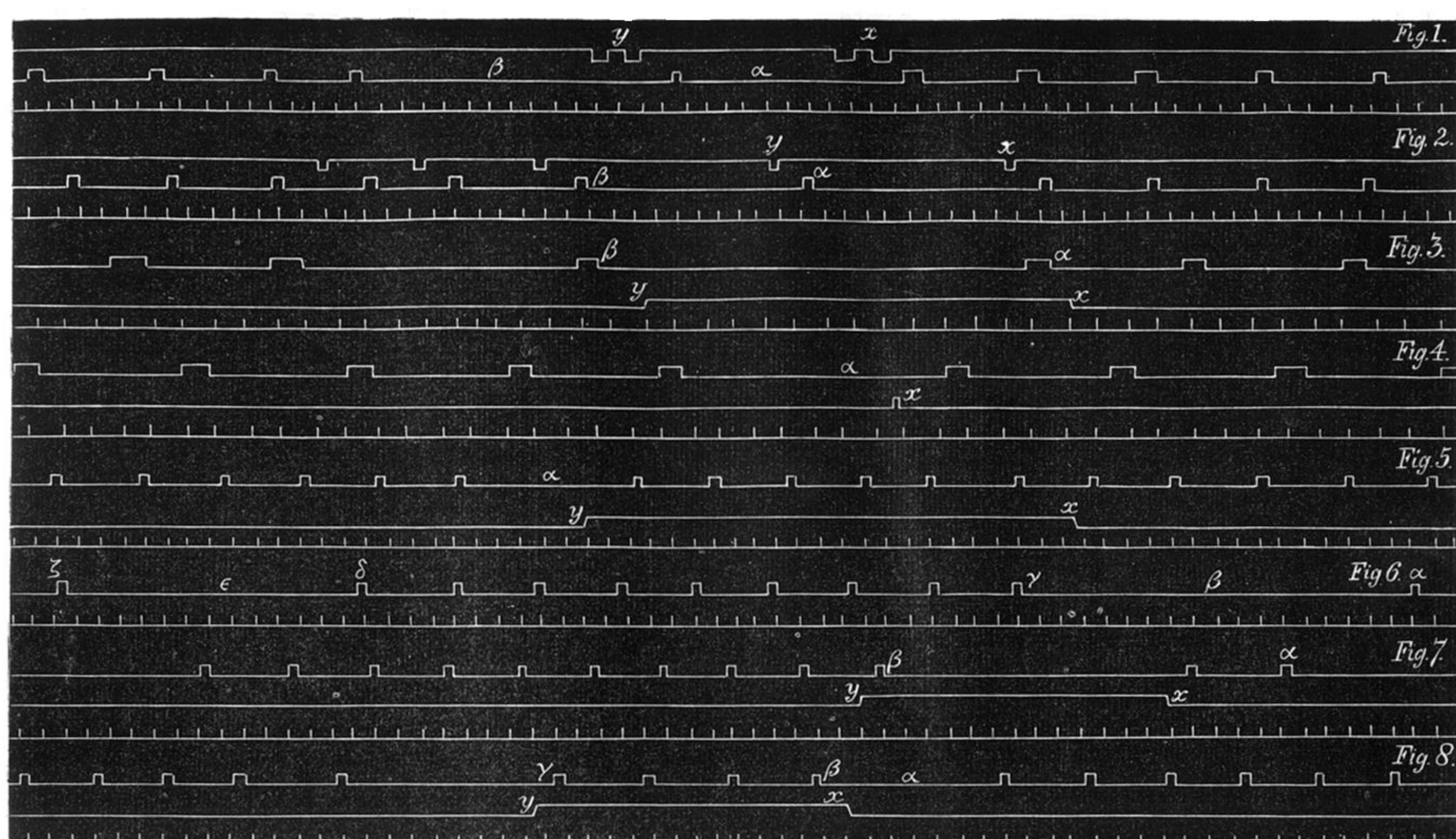


Fig. 1. Repeated single induction-shocks.

Two shocks thrown in at x causing distinct prolongation of diastole α , then followed a very feeble beat, then two more shocks at y , followed again by prolongation of diastole β . The stimulations were produced by a Du Bois-Reymond's induction-apparatus, worked by one Daniell's cell. The secondary coil at 8° . The making-shock only was used.

Fig. 2. Single induction-shock.

Single induction-shock thrown in at x , after a series of normal beats. The result was distinct inhibition for a time, then followed a feeble beat at α , then another shock was thrown in at y , followed by a pause. At β the heart commenced to beat again, and resisted the influence of the three succeeding shocks thrown in, continuing to beat normally. Making-shock only. One Daniell's cell. Coil at 5° .

Fig. 3. Constant current applied to a spontaneously beating ventricle.

The upper line indicates the beats of the ventricle, the middle line the application of the current. The kathode was placed at the aortic end of the ventricle, anode placed at the auricular end of the ventricle. No resistance in the resistance-shunt; consequently only the very smallest fraction of the current could have passed through the ventricle. Current made at x and broken at y .

The beat α started from the aortic end, the beat β from the auricular end.

Fig. 4. The same current, applied to the same heart, under exactly similar circumstances as in fig. 3, except that the break almost instantaneously followed the make. No beat was produced, but only a lengthening of the diastole α .

Fig. 5. Effect of constant current applied to the spontaneously beating heart, the auricle still attached to ventricle. Kathode at auricle, anode at aortic end of ventricle. Resistance introduced into the resistance-shunt *nil*; consequently the very feeblest current was sent through the heart. The current is made at x and broken at y . No appreciable effect during the passage of the current, except a slight quickening of the beat. The breaking of the current is marked by the prolonged diastole at α .

Fig. 6. Example of the "secondary rhythm" of the ventricle of the snail's heart.

Shows the secondary rhythm spoken of on page 334. The last of a series of 9 beats is shown at α . Then follows a pause (β) for 18 seconds; then the beats recommence at γ , going on 9 times to δ ; then again follows the pause ϵ , succeeded by another series of 9 beats, commencing at ζ .

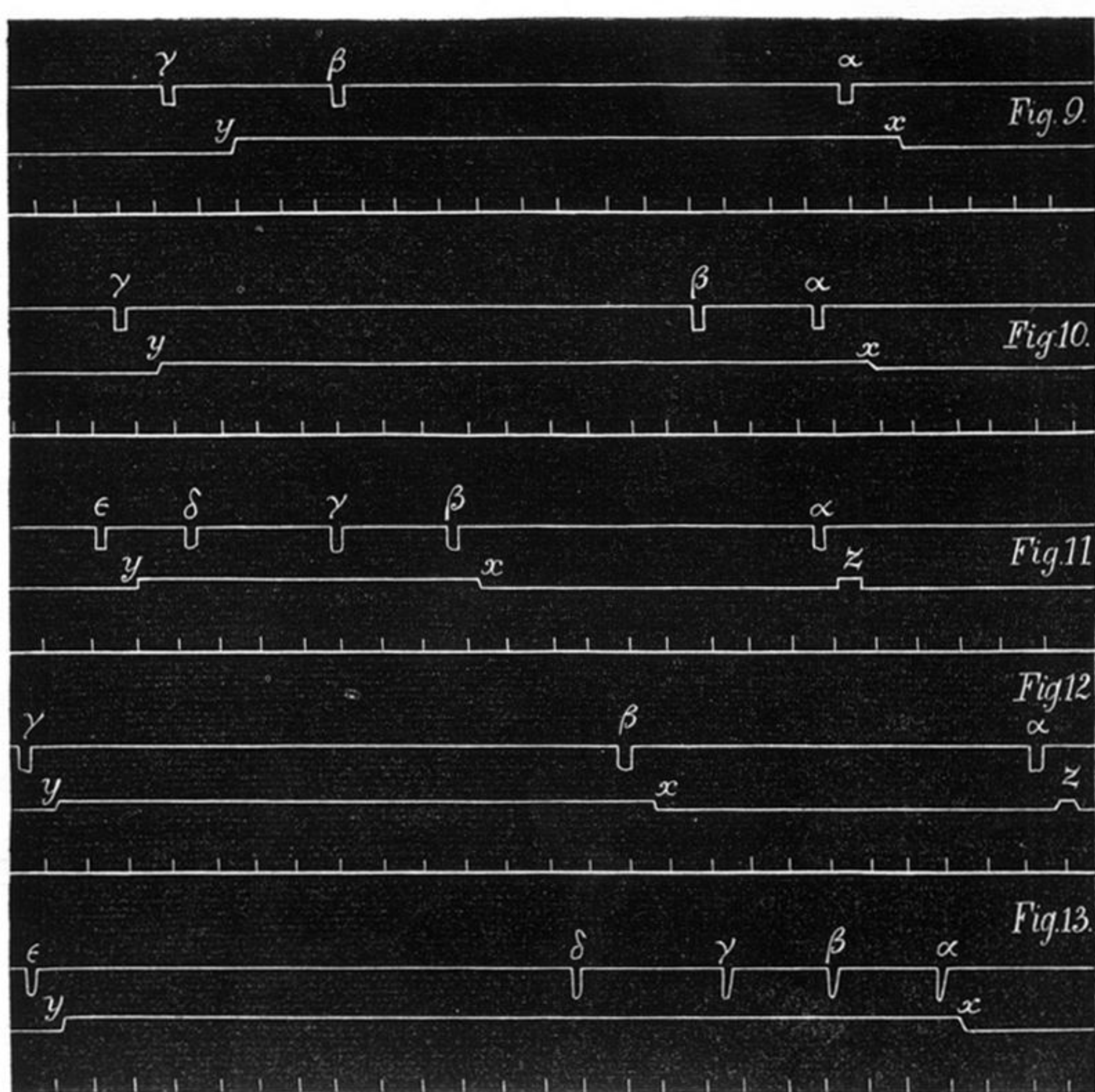
Fig. 7. Inhibition of ventricle by constant current thrown in during an active period of the secondary rhythm shown in fig. 6.

Auricle still attached to ventricle, and .1 ohm introduced into the resistance-shunt. The anode at the auricle, the kathode at the aortic end of the ventricle. An instantaneous make and break, whether thrown in during the period of rest or during the period of activity, produced no effect. α is the first of the series of beats succeeding a pause. On the making of the current at x the beats cease, but reappear at β upon the breaking of the current at y .

N.B. The point of the lever marking the ventricular beats on the upper line was not placed exactly over the point of the lever marking on the middle line the application of the current, consequently the beat β seems to precede the breaking of the current at y . In reality it succeeded it by a very short interval.

Fig. 8. Pulsation brought on by constant current thrown in during the resting-period of secondary rhythm.

Heart and strength of current same as in fig. 7. Kathode at auricle, anode at aortic end of ventricle. The resting-period or pause is seen commencing at α , but is broken by the beat β , induced by the constant current, which is made at x ; the beats continue, ceasing at γ when the current is broken at y .



The beats α , β commenced at the auricular end of the ventricle; the beat γ , due to disappearance of anelectrotonus, commenced at the aortic end of the ventricle.

Fig. 9. Constant current applied to the heart at rest.

Auricle still attached to the ventricle. Anode at the auricle, kathode at the aortic end of the ventricle. The resistance in the resistance-shunt amounted to 1 ohm. Otherwise the arrangements were the same as in fig. 12 &c. The beat α was very faint, starting from the aortic end of the ventricle. The beats β , γ started from the auricular end of the ventricle. This was an exceptional case, in which a beat (β) appeared before the breaking of the current.

Fig. 10. Constant current.

Applied to heart at rest. Kathode at auricle, anode at aortic end of ventricle.

The top line indicates beat of ventricle; the middle line indicates the application of the current, made at x and broken at y . A single Daniell's cell was used. In the resistance-shunt the resistance was 1 ohm; consequently a small fraction only of the total current passed through the heart.

Fig. 11. Same arrangement as fig. 10. Resistance in the resistance-shunt 100 ohms.

The beat at α , due to the instantaneous make and break at z , was normal in character, *i. e.* started from the auricular end of the ventricle. The beats β , γ , δ also started from the auricular end. The beat ϵ started from the aortic end of the ventricle.

Fig. 12. Constant current applied to the heart at rest.

Auricle still attached to ventricle. Anode placed at auricle, kathode at aortic end of ventricle. The whole of the current from a single Daniell's cell was sent through the heart. Otherwise the same arrangement as in figs. 10, 12, & 13.

The beat α , due to the instantaneous make and break at z , was faint, starting from the aortic end of the ventricle. β was a stronger beat, starting also from the aortic end of the ventricle. γ was a strong beat, starting from the auricular end of the ventricle.

Fig. 13. Same arrangement as figs. 10 & 11.

The whole of the current from a single Daniell's cell was thrown in. The beats α , β , γ , δ started from the auricular end, and ϵ from the aortic end of the ventricle.